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Most common artifacts are associated with factors related to the patient, to the imaging technique, image processing or problems in the apparatus[10,11]. The main patient-related artifacts are caused by motion during images acquisition and use of substances on the skin.

The present case illustrates the necessity of a strict mammographic image quality control and correlation with clinical findings for greater diagnostic accuracy. As already mentioned, the skin lesion led to simulation of a clustered pleomorphic microcalcifications which would imply the necessity of biopsy. The active quest for prevention and detection of artifacts, in association with a continued quality control of imaging, processing, storage and images analysis, reduces the incidence of misdiagnosis and costs, e should be the objective of any team involved in mammography services.

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Hypertrophic olivary degeneration secondary to central tegmental tract injury

Degeneração olivar hipertrófica secundária a lesão do trato tegmentar central

Dear Editor,

A male, 30-year-old patient presenting with a sudden-onset convergent squint attended the service complaining of diplopia. At physical examination the patient presented compromise of the left VI nerve and palatal myoclonus. Magnetic resonance imaging (Figure 1) demonstrated hypertrophic olivary degeneration (HOD) secondary to central tegmental tract injury.

Recently, the Brazilian radiological literature has been much concerned about the relevant role played by imaging methods in the improvement of the diagnosis of central nervous system diseases[1–10].

HOD is a rare phenomenon that occurs after an insult to the dentato-rubro-olivary tract (Guillain-Mollaret triangle), constituted by the dentate, rubro and inferior olivary nuclei, which are interconnected via the central tegmentar tract and superior and inferior cerebellar peduncles. This is a degenerative disorder that initially develops with hypertrophy[11]. Injury to any of such components may result in axonal interruption to the inferior olivary nucleus, leading to its degeneration[12]. In cases where the alterations are

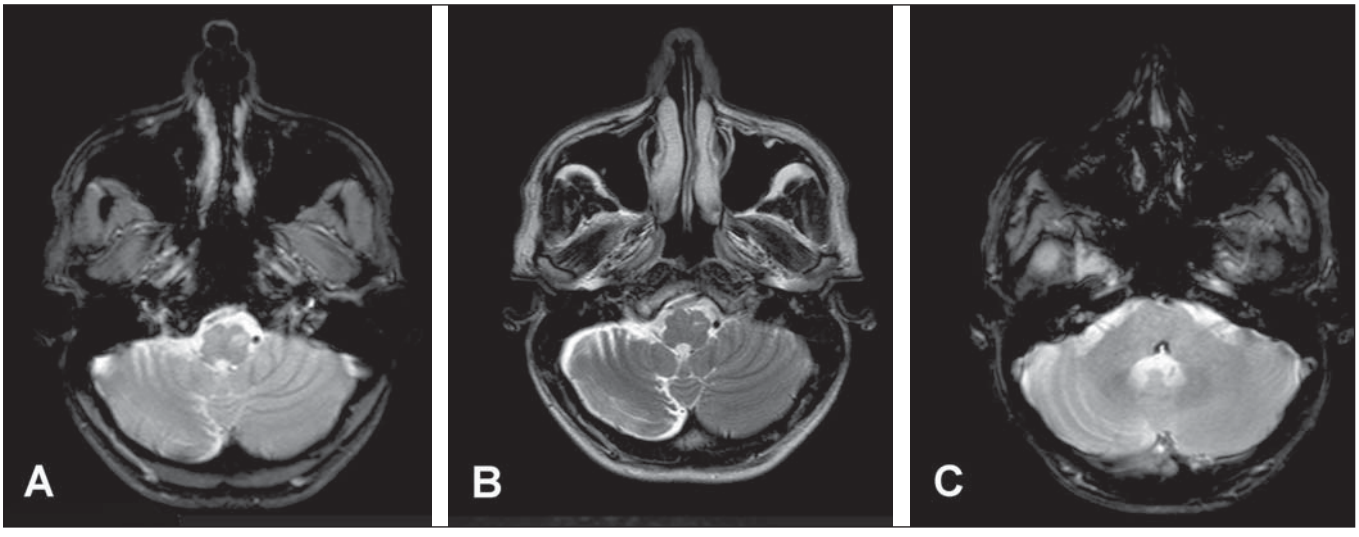

Figure 1. MRI of the brain. Axial, T2-weighted FFE image at the level of the bulb (**A**) and axial T2-weighted image (**B**) shows hypersignal in the region of the left inferior olivary nucleus, as well as accentuation of sulci in the right cerebellar hemisphere. Susceptibility-weighted imaging (**C**) at the level of the pons shows lesion with peripheral hyposignal in the pontine tegmentum (left facial colliculus), compatible with hemorrhagic focus.

restricted to the central tegmentar tract, the HOD is ipsilateral, like in the present case. In cases where there is involvement of the dentate nucleus or of the dentate nucleus or superior cerebellar peduncle, the HOD is contralateral. Bilateral compromise may be observed in cases where the lesion simultaneously affects the superior cerebellar peduncle and the contralateral central tegmentar tract, or in case of compromise of both central tegmentar tracts[11,13].

HOD is characterized as a lesion with hypersignal on T2-weighted images, in the anterolateral portion of the bulb, in the olivary region, associated with increase in the volume of such a structure that does not present contrast enhancement. Additionally, the volumetric reduction of the cerebellar hemisphere contralateral to the olivary alteration corroborates the diagnosis[14].

HOD occurs as a result of a range of insults. The symptoms are classically associated with palatal and ocular myoclonus, besides tremors. Such abnormal involuntary movements are consequential to failure of inhibition of inferior olive, since the fibers originated in the dentate nucleus are primarily inhibitory or GABAergic. In such case, expectant management is the approach to be adopted.

Macroscopic findings reveal hypertrophy instead of olivary atrophy, which is a particularity of this transsynaptic degeneration. Histopathologically, there is hypertrophy of both neurons (due to proliferation of neurofilaments and cytoplasmic vacuolization) and glial cells.

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